Indian Journal of Medical Case Reports ISSN: 2319–3832(Online)
An Open Access, Online International Journal Available at http://www.cibtech.org/jcr.htm
2016 Vol.5 (3) July-September, pp. 56-58/Pathak et al.
Case Report

CASE REPORT: HEPATIC HYDROTHORAX WITHOUT ASCITES- A RARE ENTITY

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ABSTRACT
We are presenting a case report of a 32 year old male patient, who presented to us in OPD with complaints of gradually progressive dyspnea and dry cough. In the absence of cardiopulmonary disease and significant proteinuria, the pleural effusion after investigation was diagnosed to be due to cirrhosis of liver.

Keywords: *Cirrhosis, Pleural Effusion, Dyspnoea*

INTRODUCTION
Hepatic Hydrothorax, in a cirrhotic patient is said to be when there is significant plural effusion in the absence of any primary respiratory or cardiocascular disease and it is very uncommon, hepatic hydrothorax without presence of ascites (HHAA) is a very rare complication of decompensated liver cirrhosis.
Hepatic hydrothorax is a rare differential diagnosis of a transudative pleural effusion. It is seen frequently in the presence of ascites and other features of portal hypertension due to chronic liver disease, rarely it may be the only presentation for chronic liver disease and appropriate workup for cirrhosis and causes of decompensation including hepatocellular carcinoma (HCC) needed. In this case, hepatic hydrothorax was the only manifestation of cirrhosis of liver.
Therefore, careful investigation and referral to a gastroenterologist (or hepatologist) for further work should be done in these cases.

CASE
A 32 year old male presented with complaint of gradually progressive dyspnoea and dry cough for 10 days, which was not associated with chest pain, fever, weight loss, productive cough.
He was diagnosed as a case of cirrhosis of liver with portal hypertension 4 years ago, he had undergone upper GI endoscopy which reveal`s grade II esophageal varix for which endoscopic variceal ligation was done 4 months back, and he was on regular medication since then, patient was admitted and investigations were done.

On Examination
Patient was conscious but irritable, trespiratory rate was 42 breaths (tachypnoeic) per min, blood pressure was 110/66 mm of Hg and pulse rate of 116/min, mild icterus was present, and no other finding in general examination.
On systemic examination signs of pleural effusion was present. Per abdomen examination revealed grade II splenomegaly, all other system examination was found to be normal.

Investigations
A chest radiograph was suggestive of massive Right sided plural effusion with shifting of mediastinum towards the left side (figure1)
USG Abdomen & Thorax: was also done and reveals features of cirrhosis of liver with portal hypertension with minimal fluid in peritoneal cavity with splenomegaly, in thorax suggestive of massive right sided pleural effusion.
Diagnostic as well as therapeutic thoracocentesis was done on admission and 2 litres of fluid was drained fluid was sent for investigation. It was yellowish in color but clear. Fluid was a transudative in nature

according to LIGHT'S criteria, with total protein 0.79%, sugar 172% total cell 50cells/mm^3 with 80% lymphocytes and 20% of Neutrophils, no pyogenic or acid fast bacilli were seen on smears and culture.
Blood counts were normal, Hb was 9.2gm/dl with derranged liver function test. Total bilirubin 3.33 with unconjugated bilirubin is 1.17 and conjugated bilirubin was 2.16. SGOT 154IU/L, SGPT 36IU/L, ALP 488IU/L and total protein was 6.4 with albumin 2.3 and globulin 4.1., HBsAg and antiHCV were negative. The blood sugar was 192mg/dl and renal functions were Serum creatinine was 1.42mg% and urea 62mg%. He was treated with spironolactone and uptitrated to a dose of 200 mg once daily which significantly improved and patient was discharged after 7 days on same dose of spironolactone and beta blocker, he is awaiting follow up.

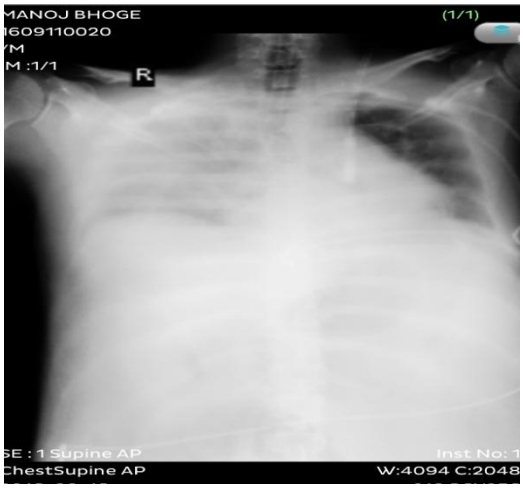

Figure 1

DISCUSSION

Hepatic hydrothorax is defined as a pleural effusion, equal to or more than 500 mL in the pleural cavity, in a patients with cirrhosis of liver without any prior cardiopulmonary disease (Strauss and Boyer, 1997). It occurs in 5–10% patients of cirrhotic patients (Cardenas *et al.,* 2004).
The exact mechanisms of pleural effusion in cirrhotics is not been well-defined. But it is thought that ascites that results from portal hypertension, crosses small diaphragmatic defects (usually <1 cm) upto pleural cavity (Zenda *et al.,* 1998) which is favoured by negative intrathoracic pressure. It is seen that, 85% of hepatic hydrothorax develops on the right side, 13% left side and 2% bilaterally.
Hepatic hydrothorax in the absence of ascites is uncommon (but some cases has been reported) (John *et al.,* 2009). Pleural effusion in patient with decompensated Cirrhosis of liver maybe asymptomatically or may be found incidentally on a chest radiograph or the patient may have difficulty in breathing associated with cough and even low oxygen levels in blood and impending respiratory failure.
Management of hepatic hydrothorax involves management of underlying chronic liver disease as well as of hepatic hydrothorax.
The management of decompensated chronic liver disease is already described by multiple international guidelines (European Association for the Study of the Liver, 2013). Screening of liver under the care of a gastroenterologist/hepatologist is recommended. Patients should be assessed for liver transplantation according to the eligibility critereas.
The management of the hydrothorax is same as of ascites. In the hydrothorax a combination of low salt, high protein diet, and diuretics. Aldosterone antagonists and loop diuretics can be used in isolation or in combination.
Symptomatic pleural effusions may require therapeutic thoracocentesis. For recurrent pleural effusions chest drainage with pleurodesis can be used, which has been described (Falchuk *et al.,* 1977; Lin *et al.,* 2000).

Rapid reaccumulation of fluid from increased portal pressure (Ikard and Sawyers, 1980; Rodriguez-Panadero and Antony, 1997) is common. In dwelling pleural catheters are being used in the malignant pleural effusions and in those who are unfit for specific hepatic intervention.

Transjugular intrahepatic portosystemic shunt (TIPSS) is used in refractory cases of hydrothorax and in patients who do not tolerate diuretics (Siegerstetter *et al.*, 2001). It acts to decrease portal pressure which favours formation of ascites. Studies shows TIPSS success response rates ranging between 70% and 80% (Gordan *et al.*, 1997; Siegerstetter, 2001).

Conclusion

Hepatic hydrothorax is a rare complication in decompensated liver disease patients and may be the only presentation in patients of cirrhosis of liver.

Management is similar as that of ascites includes sodium restriction, and oral diuretics.

Chest drain may be required in some cases.

Transjugular intrahepatic portosystemic shunt is a definitive and effective treatment modality for recurrent hepatic hydrothorax.

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